Ungar, Susan

To: STIC-ILL
Subject: Paper for Exmination of 09/755,233

Hi

I need the following for examination of SN 09/755,233

1. Kobayashi et al, Gastroenterology, 1990, Vol 98, No. 5, Part 2), A289

2. Kobayashi et al (J. Immunol., 1989, 143(8)2567-2574.

Thanks
Susan Ungar
1642
703-305-2181
CM1-8B05

I TUMOR-NECROSIS-FACTOR-ALPHA DECREASES EXPRESSION OF THE INTESTINAL IGG FC BINDING-SITE BY HT29-N2 CELLS

AU HAMADA Y; **KOBAYASHI K**; BROWN W R (Reprint)
CS VET AFFAIRS MED CTR, GASTROENTEROL SECT, 111E, 1055 CLERMONT ST, DENVER, CO, 80220; UNIV COLORADO, SCH MED, DENVER, CO, 80202
CYA USA

SO IMMUNOLOGY, (1991) Vol. 74, No. 2, pp. 298-303.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 17

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Previously, we describe a unique binding site for the Fc region of IgG in human intestinal goblet cells, but regulation of the intestinal IgG Fc binding site (Fc-gamma-IBS) has not been clarified. In this work, we examined the effects of tumour necrosis-alpha (TNF-alpha) and interferon gamma (IFN-gamma) on expression of the Fc-gamma-IBS in HT29-N2 colonic cancer cells, which differentiate readily into goblet cells containing the binding site when grown in galactose-containing medium. Expression of the site was monitored immunocytochemically and by ELISA on homogenates of the cells. TNF-alpha in doses of 0.1-100 ng/ml caused a reduction in expression of the Fc-gamma-IBS and the proportion of cells positive for mucin (as demonstrated by Alcian blue stain), without affecting the viability of the cells. The effects of TNF-alpha on the Fc-gamma-IBS and mucin production could not be attributed to a decreased proliferative rate of the cells, as the cells' incorporation of 5-bromo-2'-deoxyuridine was unaffected. By contrast with TNF-alpha, IFN-gamma (i) did not affect the proportion of cells expressing the Fc-gamma-IBS, (ii) decreased the viability of the cells, and (iii) increased cell proliferation. Additional evidence of specificity of the TNF-alpha effect on the Fc-gamma-IBS was that TNF-alpha did not affect expression of the polymeric immunoglobulin receptor (secretory component), whereas IFN-gamma increased it. We conclude that TNF-alpha may suppress expression of the Fc-gamma-IBS by **colonocytes** and oppose differentiation of the cells towards mucin-producing cells.

ANSWER 1 OF 1 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1990:298776 BIOSIS
DN BR39:16957
TI EXPRESSION OF AN IGG FC BINDING SITE **I-FCBS** BY CULTURED **COLONOCYTES** AND COLONIC TUMORS.
AU **KOBAYASHI K**; MIZUNO Y; HIBI T; TSUCHIYA M; HAMADA Y; BROWN W R
CS DEP. INTERN. MED., ICHIKAWA GENERAL HOSP., TOKYO.
SO 91ST ANNUAL MEETING OF THE AMERICAN GASTROENTEROLOGICAL ASSOCIATION AND DIGESTIVE DISEASE WEEK, SAN ANTONIO, TEXAS, USA, MAY 12-18, 1990. GASTROENTEROLOGY. (1990) 98 (5 PART 2), A289.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference
FS BR; OLD
LA English
CC General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Biophysics - Membrane Phenomena 10508
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Digestive System - Pathology *14006
Neoplasms and Neoplastic Agents - Immunology *24003
Neoplasms and Neoplastic Agents - Biochemistry *24006
Developmental Biology - Embryology - Morphogenesis, General *25508
Immunology and Immunochemistry - General; Methods *34502
BC Hominidae 86215
IT Miscellaneous Descriptors
ABSTRACT HUMAN COLON CARCINOMA HT29 CELLS HT29N2 CELLS COLO 205 CELLS LOVO CELLS IMMUNOGLOBULIN G MUCIN PRODUCTION CELL DIFFERENTIATION

3 ANSWER 6 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1990:298776 BIOSIS
DN BR39:16957
TI EXPRESSION OF AN IGG FC BINDING SITE I-FCBS BY CULTURED **COLONOCYTES** AND COLONIC TUMORS.
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CODEN: GASTAB. ISSN: 0016-5085.
DT Conference
FS BR; OLD
LA English

ANSWER 8 OF 23 PASCAL COPYRIGHT 2003 INIST-CNRS. ALL RIGHTS RESERVED.
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AN 2003-0290256 PASCAL

TIEN Coprocytobiology: On the nature of cellular elements from stools in the pathophysiology of colonic disease
Defining the pathologic and clinical significance of dysplasia and metaplasia in the gastrointestinal tract

AU NAIR Padmanabhan; LAGERHOLM Sara; DUTTA Sudhir; SHAMI Samina; DAVIS Kirk; MA Shuzhen; MALAYERI Mehran
FLOCH Martin H. (ed.); WEST A. Brian (ed.); NAIR Saraswathi (ed.)

CS NonInvasive Technologies, Columbia, United States; Division of Human Nutrition, Department of International Health, Johns Hopkins University, Baltimore, United States; Division of Gastroenterology, Department of Medicine, Sinai Hospital of Baltimore, Baltimore, Maryland, United States
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SO Journal of clinical gastroenterology, (2003), 36(5, SUP), S84-S93, 80 refs.
Conference: Yale University School of Medicine Workshop, Norwalk, CT (United States), 4 Oct 2002
ISSN: 0192-0790 CODEN: JCGADC

DT Journal; Conference

BL Analytic

CY United States

LA English

AV INIST-18331, 354000117982130120

AB The gastrointestinal epithelium is known to undergo constant and rapid renewal resulting in millions of cells being shed into the fecal stream every day. The conventional wisdom was that these cells disintegrate upon exfoliation and will not survive the transit through the intestinal tract. In 1990, we (P.N.) made the discovery that a significant number of these cells remain intact and viable and that they can be isolated. The implications of this important discovery became apparent when we demonstrated that these cells are exclusively of colonic origin, are anatomically representative of the entire colon, and can be used for clinical investigations of disease processes. The term coprocytobiology (CCB) was coined to encompass the broad range of applications of this new technology. The somatic cell sampling and recovery (SCSR) process involves the isolation of exfoliated **colonocytes** from a small sample of stool (1 g) collected and transported in a unique medium at ambient temperature, providing cells for the detection of a number of biomarkers of disease propensity. These exfoliated **colonocytes** express cytokeratins indicating epithelial lineage as well as colonspecific antigen. Over the years, the study of exfoliated **colonocytes** has provided striking new insights into the biology of colon cancer and inflammatory bowel disease, including detection of p53 gene mutations, reverse transcriptase polymerase chain reaction amplification, and identification of CD44 splice variants, neoplasia-associated specific binding of plant lectins, and expression of COX-2, the inducible form of cyclooxygenase. The functional diversity of cells isolated by SCSR is revealed by the demonstration of cell surface markers such as secretory component, **IgA**, and IgG on the one hand and the amplification and cloning of the human insulin receptor and the expression of the multidrug resistance gene mdr-1 on the other hand. This review portrays the immense potential of CCB as a powerful tool for investigating the pathophysiology of disease, identifying genetic variants in pharmacogenetics, assessment of mucosal immunity, and several other applications that use somatic cells.

ANSWER 23 OF 23 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 10
AN 1990:4469 BIOSIS
DN BA89:4469
TI IDENTIFICATION OF A UNIQUE IGG FC BINDING SITE IN HUMAN INTESTINAL EPITHELIUM.
AU KOBAYASHI K; BLASER M J; BROWN W R
CS GASTROENTEROL. DIV. 111E, VETERANS ADM. MED. CENTER, 1055 CLERMONT ST., DENVER, CO 80220.
SO J IMMUNOL, (1989) 143 (8), 2567-2574.
CODEN: JOIMA3. ISSN: 0022-1767.
FS BA; OLD
LA English
AB In experiments to determine whether serum antibodies in patients with Crohn's disease could be used as probes for detecting potentially etiologic Ag in the patients' tissues, we found that peroxidase (HRP)-labeled IgG from healthy persons, as well as from the patients, bound to normal colonic and small intestinal epithelium, mostly or entirely to goblet cells. The binding was due to a reaction involving the Fc region of IgG because HRP-labeled Fc fragments of IgG bound, but HRP-Fab, HRP-**IgA**, and HRP-bovine albumin did not, and because binding of HRP-IgG was inhibited competitively by unlabeled IgG or Fc fragments but not by IgG Fab fragments or **IgA**. These immunohistochemical results were confirmed by ELISA with microtiter wells coated with a sonicated homogenate from human **colonocytes**. The epithelial IgG Fc binding site was characterized by SDS-PAGE as consisting of a high Mr (> 200,000 Da) and a 78,000-Da component. It bound all four subclasses of human IgG and bound aggregated as well as monomeric IgG. It is distinct from known human Fc-.gamma.R by lack of recognition by mAb to those receptors and differences in affinity for various subclasses of human and murine IgG. This unique IgG Fc binding site might be involved in immunologic defense of the gut, perhaps by mediating reactions between foreign Ag and the contents of goblet cells.